International Journal of Sport Nutrition, 1995, **5**, S74-S83

Micronutrients (Magnesium, Zinc, and Copper): Are Mineral Supplements Needed for Athletes?

Henry C. Lukaski

Mineral elements, including magnesium, zinc, and copper, are required by the body in modest amounts for the maintenance of health and for the development of optimal physiological function. For athletes, adequate amounts of these minerals are required for physical training and performance. Studies of athletes during training, as compared to nontraining control subjects, indicate the potential for increased losses of minerals in sweat and urine. Some studies report suboptimal intakes of minerals, particularly among athletes who are actively attempting to lose weight to meet standards for competition. However, most athletes consume diets that provide adequate amounts of minerals to meet population standards. Athletes should be counseled to consume foods with high nutrient density rather than to rely on mineral supplements. General use of mineral supplements can alter physiological function and impair health.

As athletes seek to gain a competitive edge in performance, they search for technical, psychological, and biological advantages. One factor that is actively investigated is diet. Thus, athletes have become conscious of the benefits of proper eating practices and dietary components that support intensive physical training and promote optimal performance. One area of nutrition in which athletes, both recreational and competitive, have become increasingly interested is the beneficial role that adequate mineral element nutrition can have in physical performance.

It is clear that many of the physiological and biochemical functions of magnesium, zinc, and copper are important for energy metabolism and hence physical performance. In general, these minerals act as inorganic regulators of various aspects of energy production and utilization. Following are established roles of minerals in the development of optimal physical performance:

Energy metabolism

- Glycolysis
- Lipolysis
- Proteolysis
- Phosphagen hydrolysis

Biochemical functions

- Enzymatic activity
- Structural component
- Catalytic site component
- Antioxidant defense

Magnesium, zinc, and copper may be needed to maintain the structural integrity of essential molecules or may be required at the catalytic site of an enzyme to enable the enzyme to function properly. Thus, mineral elements have the potential to influence physical performance.

Henry C. Lukaski is with the USDA Agriculture Research Service, Grand Forks Human Nutrition Research Center, Box 9034 University Station, Grand Forks, ND 58202-9034.

Assessment of Mineral Nutritional Status

Severe deficiencies of magnesium, zinc, or copper in the U.S. population are rare. These abnormalities generally are restricted to individuals with genetic disturbances of mineral metabolism or are secondary to another metabolic or pathological disturbance. However, marginal deficiency associated with prolonged dietary restriction of mineral intake may be common in the U.S. Thus, marginal deficiencies of magnesium, zinc, and copper may be seen in the otherwise healthy athletic population.

Dietary Mineral Intake

A basic approach for the assessment of nutritional status is to evaluate the adequacy of daily nutrient intake (Table 1). By using dietary records and an appropriate nutrient database, one can estimate the daily nutrient intake of an individual. Nutritional adequacy may be examined in relation to a population standard. For example, individual mineral intakes are compared to the recommended daily allowance (RDA) for magnesium and zinc or the estimated safe and adequate daily dietary intake (ESADDI) for copper. Values from 70 to 100% of the population standard are considered adequate for an individual.

Blood Biochemical Markers of Mineral Status

Because minerals play a fundamental role in cellular energy metabolism, it is important to determine tissue mineral concentrations or to obtain a functional assessment of cellular mineral status. Unfortunately, such direct measurement in tissues or cells is impractical, so investigators rely on indirect assessments of mineral nutritional status in tissues (Table 1).

One experimental approach is to determine the concentration of a mineral in blood and then to compare the individual value with the range of normal values established

Table 1 Experimental Approaches for Assessment of Human Mineral Nutritional Status

Approach	Variable	Standard
Dietary records	Daily intake	>70% RDA or ESADDI
Blood biochemical index	Plasma/serum concentration; mineral-specific enzyme activity in erythrocytes, platelets, or leukocytes	Within range of values for nutritionally adequate, gender-matched, control subjects
Excretion	Urinary losses	Increased absolute (mg/day) or relative (% daily intake) amount in timed urine output

Note. RDA = recommended dietary allowance; ESADDI = established safe and adequate daily dietary intake.

in nutritionally adequate, age- and gender-matched subjects. Although this approach is used for assessment of plasma or serum concentration of magnesium, zinc, and copper, the sensitivity of this method to detect marginal or mild deficiency states is inadequate (29, 31). Another strategy is measurement of erythrocyte concentrations of these minerals. Unfortunately, this approach also is an insensitive indicator of marginal mineral deficiency states of humans. A final investigational technique for nutritional assessment is the measurement of the activity of a specific mineral-containing enzyme. Although several potential candidates are available, this approach either has not been implemented fully or has been hampered by the need to measure isoenzymes that are mineral-specific.

Effects of Exercise on Mineral Losses

In addition to biochemical assessment of tissue mineral status, examination of changes in daily losses of minerals in response to exercise may provide evidence of increased mineral needs. Two routes of mineral excretion include sweat and urine. Mineral losses by both of these sources are increased during prolonged exercise and may represent significant factors in the perturbation of mineral balance in the body if compensation with adequate dietary mineral intake is not accomplished.

Magnesium

In the body, magnesium is a ubiquitous element that plays a role in a wide range of fundamental cellular reactions. Magnesium is involved in more than 300 enzymatic reactions in which food is metabolized and new products are formed (29). Some of these activities include the glycolytic pathway, fat and protein metabolism, adenosine triphosphate (ATP) hydrolysis, and the second-messenger system. In addition, magnesium acts as a physiological regulator of membrane stability and in neuromuscular, cardiovascular, immune, and hormonal function.

Physical Activity and Magnesium Status

A clinical report (23) suggested that magnesium deficiency can impair physical performance. A young, adult female tennis player reported frequent episodes of muscle spasms associated with prolonged outdoor exercise. A diagnosis of magnesium deficiency was based on hypomagnesemia (serum magnesium: 0.65 mmol/L; normal range: 0.8–1.20 mmol/L) in the presence of otherwise normal physical, neurological, and blood biochemical examinations. Daily treatment with 500 mg of magnesium gluconate relieved the muscle spasms within a few days.

Dietary surveys of male and female athletes indicated an interesting pattern of magnesium intake. Generally, magnesium intakes for male athletes equaled or exceeded the RDA of 350 mg, but those of female athletes were about 60–65% of the RDA of 280 mg (25). Regardless of gender, athletes participating in wrestling and other sports with weight restrictions for competition or in sports requiring an aesthetic component (e.g., ballet or gymnastics) tended to consume inadequate amounts of dietary magnesium, 30–55% of the RDA (18).

Other cross-sectional studies of athletes have not confirmed an inadequate magnesium intake. Comparisons of dietary magnesium among male and female cross-country skiers and control subjects found magnesium intakes of 170–185% RDA for the athletes, as compared to 108–116% for the gender-matched controls (11).

Fogelholm et al. (12) found no adverse effect of physical training on dietary magnesium and blood biochemical indices of magnesium status among athletes and nontraining control subjects. Plasma and erythrocyte magnesium concentrations were similar among the two groups. Dietary magnesium, however, was greater among the athletes than the control subjects (548 vs. 436 mg/day). These findings indicate that when athletes consume diets containing amounts of magnesium that meet or exceed established population norms, blood biochemical indices of magnesium status are not impaired.

Redistribution and increased loss of magnesium from the body have been observed during and immediately after a bout of exercise. Following exercise (compared to preexercise conditions) a shift in magnesium from the plasma into the erythrocytes was found (7). The direction and magnitude of this transient redistribution of magnesium were dependent on the intensity of the preceding exercise; the more anaerobic the exercise, the greater the movement of magnesium from plasma into erythrocytes. In addition, urinary excretion of magnesium increased 21% on the day of exercise, as compared to control or nonexercise values, and returned to nonexercise levels on the day following exercise (7, 33). These findings suggest a temporary increase in magnesium loss following exercise that returns to equilibrium in the subsequent days if additional exercise is not performed. Importantly, the amount of magnesium lost in the urine was related to the degree of exercise-induced anaerobiosis, indexed by postexercise oxygen uptake and plasma lactate concentration (7). This relationship has been interpreted to suggest an increased need for magnesium when glycolytic metabolism is dominant.

Sweat losses of magnesium increase during exercise (4). Men performing controlled ergocycle exercise in the heat (100 °F) lost 15.2–17.8 mg magnesium per day in sweat. This output represented 10–15% of total daily magnesium losses (feces, urine, and sweat) and 4.4–5.2% of daily magnesium intake.

Magnesium Supplementation and Performance

Supplementation of the diets of competitive athletes with magnesium salts has been reported to improve cellular metabolism (13). One approach has been to examine the physiological effects of supplementation with a magnesium salt or a placebo. Among competitive female athletes with plasma magnesium concentrations at the low end of the range of normal values, serum total creatine kinase and creatine kinase isoenzyme MB decreased after training in the women supplemented daily for 3 weeks with 360 mg magnesium as magnesium aspartate, as compared to the women receiving placebo (14). During an exhaustive rowing-ergometer test, serum lactate concentration and oxygen consumption were decreased in male competitive rowers supplemented with magnesium (360 mg/day) for 4 weeks, as compared to rowers receiving a placebo (13). These results suggest a potentially beneficial effect of magnesium on energy metabolism and work efficiency.

Magnesium supplementation also has been implemented to increase muscle strength and power (3). Young men participating in a 7-week strength training program received either a placebo or a magnesium supplement (magnesium oxide) to attain a daily intake of 8 mg/kg body weight. Magnesium intakes were estimated to be 250 and 507 mg/day for the placebo and supplement groups, respectively. The groups had similar strength of the quadriceps muscles before treatment. Peak knee-extension torque increased significantly in the magnesium-supplemented group, which suggested a role for magnesium in activities requiring a dominant contribution of glycolytic metabolism.

Important questions surround the results of these magnesium supplementation trials. It would be useful to know the athletes' magnesium status, either in terms of usual magnesium intake or some blood biochemical indices of magnesium nutriture, before and after supplementation. This important question must be addressed if the role of supplemental magnesium is to be described as a nutritional or a pharmacological agent. In addition, it is unclear whether magnesium or aspartate is the agent responsible for reported effects on metabolism.

Zinc

This mineral element is essential for the function of more than 300 enzymes from many species (34). Zinc-containing enzymes participate in carbohydrate, lipid, protein, and nucleic acid metabolism. In addition, some zinc-dependent enzymes, such as lactate dehydrogenase and carbonic anhydrase, play critical roles in exercise metabolism by regulating glycolysis and carbon dioxide removal. Zinc also is required for the structural integrity of the antioxidant enzyme, superoxide dismutase.

Physical Activity and Zinc Status

Interest in the interaction between physical activity and zinc nutritional status came from the observation that some endurance runners had significantly decreased serum zinc concentrations in comparison to sedentary men (9). Twenty-three percent of the runners had zinc concentrations less than 11.5 μmol/L, the lower limit of the range of normal serum zinc values. Also, serum zinc concentration was inversely related to training mileage. In a survey of elite German athletes, no significant difference in serum zinc concentrations was found between athletes and sex-matched control subjects (16). However, some of the female and the male athletes had hypozincemia (<12 μmol/L). Dietary zinc was not reported. Among female marathon runners, plasma zinc concentrations were at the lower limit of the range of normal values, but 22% of the values were less than the lower limit (30). In contrast, no differences in plasma zinc concentrations were found between groups of male collegiate athletes and nonathletic controls (26). Whether these differences in circulating zinc are attributable to inadequate dietary zinc or other factors relating to physical activity cannot be determined.

Studies including parallel measurements of dietary zinc and circulating zinc provide some insight regarding the interaction between zinc intake and plasma zinc in physically active individuals. In a sample of female long-distance runners, mean plasma zinc was within the range of normal values (6). However, 29% of the women had hypozincemia, and 50% consumed less than the daily recommended intake for zinc, 12 mg zinc per day. Also, mean plasma zinc concentrations were similar for a group of collegiate female swimmers and nonathletic female controls; dietary zinc was less than the RDA for about 15% of both the athletes and the control subjects (27).

Serum zinc concentrations were decreased significantly in six male long-distance runners studied longitudinally during a training period of more than 9 months (5). The observed decrease was after 5 months of intensive training. Among the principal zinc-transport proteins, serum concentrations of albumin increased significantly, but alpha-2 macroglobulin did not change, in parallel with the observed decrease in serum zinc concentration. Further, the ratio of zinc to protein (albumin plus alpha-2 macroglobulin) decreased about 30% after 5 months of training. This decrease could not be explained

by concomitant increases in plasma volume or zinc-carrier proteins, both of which would be expected to decrease serum zinc concentrations and the ratio of zinc to protein.

Two hypotheses may explain the serum zinc data. It may be that the decreased serum zinc was the result of increased degradation of muscle and subsequent increased urinary zinc excretion. Couzy et al. (5) reported a 25% increase in serum myoglobin concentration in parallel with decreased serum zinc. Unfortunately, no data on urinary zinc loss were presented. An alternative hypothesis is a decrease in daily zinc intake. Estimates of zinc intake were not provided.

The effects of exercise on body losses of zinc have been estimated. Studies consistently indicate significant increases in urinary zinc excretion (about 50%) on the day of strenuous exercise as compared to the preceding nonexercise day (1, 6). The magnitude of the increase in urinary zinc loss associated with strenuous running has been estimated to be approximately 1 mg/day, or about 6% of the recommended daily zinc intake.

In general, sweat losses of trace minerals are difficult to measure because of analytical challenges (i.e., very low concentrations of minerals) and technical problems with collection of whole-body sweat. In addition, regional differences in sweat mineral concentrations occur (2, 4). Whole-body surface zinc loss under conditions of no exercise has been estimated to be about 0.8 mg/day (21). Assuming a sweat loss of 2 L and a whole-body sweat concentration of 600 μg/L, surface loss of zinc during exercise may reach 1.5 mg. Thus, surface zinc loss may approach 10% of the recommended daily intake of zinc.

Zinc Supplementation and Performance

The effect of zinc supplementation on human performance has been reported only for muscle strength and endurance (22). Sixteen middle-aged women received a zinc supplement (135 mg/day) and a placebo in a double-blind, crossover design study. Muscle strength and endurance were measured with an isokinetic, one-leg exercise test by using a standardized dynamometer protocol before and after 14 days of experimental treatment. As compared to placebo, zinc supplementation significantly increased dynamic isokinetic strength at 180°/s of angular velocity and isometric endurance. Because these types of muscular strength and endurance require recruitment of fast-twitch glycolytic muscle fibers, it was hypothesized that zinc may be required in muscular work that relies predominantly on glycolysis with high lactate production. Because neither the zinc status nor changes in zinc excretion in response to physical training of the participants were described, it is unclear whether the amount of the zinc supplement (135 mg/day) exerted a nutritional or pharmacological effect on performance. Furthermore, the amount of the supplemental zinc should be considered hazardous because of potential adverse effects on health (10, 19, 32, 35).

Copper

As compared to magnesium and zinc, which contribute about 19 g and 2.3 g, respectively, to the body of a 70-kg man, copper provides only 72 mg (20). Nevertheless, copper is a critical nutrient involved in many aspects of energy metabolism (28). Copper is an important component of hemoglobin, myoglobin, and the cytochromes and is needed for proper utilization of iron, for catecholamine metabolism, and for protection against damage to cells caused by oxidative damage. This antioxidant effect is regulated by the action of a cytosolic enzyme, superoxide dismutase.

Physical Activity and Copper Status

Limited research has been conducted relating copper nutritional status to human physical performance. Cross-sectional comparisons of athletes and control subjects not involved in physical training found no differences in plasma or serum copper concentrations (9, 17, 26). Although not statistically different, plasma/serum copper concentrations of the athletes were slightly greater (3–4%) than those of the nontraining controls.

Awareness of a potential adverse effect of physical activity on copper status began with an abstract describing decreases in blood biochemical indices of copper nutriture among swimmers who increased their training (8). Both serum copper and ceruloplasmin concentrations decreased significantly from before to the end of a competitive collegiate swimming season. In contrast, another report indicated no changes in either plasma copper or ceruloplasmin concentrations in collegiate swimmers during the competitive season (27). Importantly, more than 90% of the swimmers consumed adequate amounts of dietary copper (>1 mg/day) in the latter study (27).

Because of the catalytic role of copper in the metalloenzyme superoxide dismutase, a potent antioxidant, the interaction of dietary copper and physical training was examined in a longitudinal study (27). As compared to nontraining, age- and sex-matched control subjects, female and male athletes had significant increases in superoxide dismutase activity in erythrocytes during physical training. Because dietary copper was within established guidelines, chronic physical training apparently stimulates a protective mechanism for the control of potentially damaging free radicals. It remains to be demonstrated that inadequate copper intake inhibits this protective response during chronic physical training.

Mineral Nutriture and Prediction of Performance

Because of the role of minerals in various aspects of energy metabolism, it is reasonable to suggest that indices of magnesium, zinc, and copper nutritional status might be related to physical performance. In a study of collegiate swimmers, nutrient intake and blood biochemical indicators of nutritional status were assessed and used to predict 100-yard freestyle times at different times during the competitive season (24). As expected, biochemical indices of iron status (transferrin saturation, serum ferritin, and plasma iron) were significant predictors of swimming performance. In addition, erythrocyte magnesium concentration, superoxide dismutase activity, and daily intakes of copper, iron, magnesium, and zinc also were significant independent predictors of swim performance. The derived models for predicting 100-yard swim times had impressive coefficients of determination and acceptable standard errors (R^2 = .93 and .95; *SEE* = 0.8 s and 1.2 s for male and female swimmers, respectively). This preliminary observation supports the hypothesis that mineral nutriture is a potentially important factor in physical performance.

Adverse Effects of Mineral Supplement Use

The general use of mineral supplements is not recommended unless under the guidance of a physician or a registered dietitian. This guideline is presented because indiscriminate use of mineral supplements can lead to adverse nutritional and health effects that are dependent on the amount of the supplement and the duration of its use (Table 2).

Ingestion of magnesium supplements in amounts exceeding 500 mg/day often results in gastrointestinal disturbances, particularly diarrhea in some individuals, and

Table 2 Adverse Effects of Indiscriminate Mineral Supplementation

Mineral	Amount	RDA or ESADDI	Consequence
Magnesium	500 mg/day	280[a]–350[b] mg/day	GI disturbances
Zinc	50 mg/day	12[a]–15[b] mg/day	Copper deficiency
	>20 mg/day		Decrease HDL cholesterol
Copper	10–15 mg/day	1.5–3.0 mg/day	GI distress
	>20 mg/day		Toxicity

Note. GI = gastrointestinal.
[a]Female RDA. [b]Male RDA.

may exert a negative effect on phosphate balance (32). As with magnesium, supplemental zinc may elicit some significant metabolic aberrations. Excessive zinc supplementation can inhibit absorption of copper from the diet, even when the supplement is taken independently of meals (35). Ingestion of zinc supplements in excess of 50 mg/day can induce copper deficiency in humans (10). Zinc supplements of 160 mg/day taken for 16 weeks have been reported to significantly decrease high-density lipoprotein cholesterol (HDL) concentrations (19). It has been suggested that the use of zinc supplements ranging from 17 to 50 mg/day is sufficient to prevent an exercise-induced increase in HDL concentration (15). It has been recommended, however, that if zinc supplements are used, the zinc consumed should not exceed 15 mg/day (15).

Because of copper's capacity for participation in oxidative processes, routine ingestion of large amounts of copper in supplement form is not recommended. Toxicity of copper for humans has been estimated at intakes exceeding 20 mg/day (28). Ingestion of 10–15 mg of inorganic copper can cause nausea, vomiting, and diarrhea. In larger doses, intravascular hemolysis may occur.

Supplementation and Performance

The effect of vitamin and mineral supplementation on performance has been examined. Thirty male, trained, long-distance runners participated in a 9-month, crossover-design experiment in which supplements or placebos were ingested for 3 months, followed by a 3-month period during which no experimental treatment was given; then the treatments were reversed for the final 3 months (36, 37). On the basis of laboratory and field performance tests, there was no measurable ergogenic effect of multivitamin and mineral supplementation. Analysis of dietary records indicated that nutrient intakes, exclusive of supplements, were at least 70% of RDA or ESADDI values. Blood biochemical measurements of nutritional status were within ranges of normal values. These findings support the hypothesis that there is no beneficial effect of nutritional supplements on performance when athletes consume diets adequate in essential nutrients.

References

1. Anderson, R.A., M.M. Polansky, and N.A. Bryden. Acute effects on chromium, copper, zinc, and selected variables in urine and serum of male runners. *Biol. Trace Elem. Res.* 6:327-336, 1984.

2. Aruoma, O.I., T. Reilly, D. MacLaren, and B. Halliwell. Iron, copper and zinc concentrations in human sweat and plasma: The effect of exercise. *Clin. Chim. Acta* 177:81-90, 1988.
3. Brilla, L.R., and T.F. Haley. Effect of magnesium supplementation on strength training in humans. *J. Am. Coll. Nutr.* 11:326-329, 1992.
4. Consolazio, C.F., L.O. Matoush, R.A. Nelson, R.S. Harding, and J.E. Canham. Excretion of sodium, potassium, magnesium, and iron in human sweat and the relation of each to balance and requirements. *J. Nutr.* 79:407-415, 1963.
5. Couzy, F., P. Lafarague, and C.Y. Guezennec. Zinc metabolism in the athlete: Influence of training, nutrition and other factors. *Int. J. Sports Med.* 11:263-266, 1990.
6. Deuster, P.A., B.A. Day, A. Singh, L. Douglass, and P.B. Moser-Veillon. Zinc status of highly trained women runners and untrained women. *Am. J. Clin. Nutr.* 49:1295-1301, 1989.
7. Deuster, P.A., E. Dolev, S.B. Kyle, R.A. Anderson, and E.B. Schoomaker. Magnesium homeostasis during high-intensity anaerobic exercise in men. *J. Appl. Physiol.* 62:545-550, 1987.
8. Dowdy, R.P., and J.R. Burt. Effect of intensive, long-term training on copper and iron nutriture in man. *Fed. Proc.* 39:786, 1980.
9. Dressendorfer, R.H., and R. Sockolov. Hypozincemia in runners. *Phys. Sportsmed.* 8:97-100, 1980.
10. Fischer, P.W.F., A. Giroux, and M.R. L'Abbe. Effect of zinc supplementation on copper status in adult man. *Am. J. Clin. Nutr.* 40:743-746, 1984.
11. Fogelholm, M., J.-J. Himberg, K. Alopaeus, C.-J. Gref, J.T. Laakso, J. Lehto, and H. Mussalo-Ruahamaa. Dietary and biochemical indices of nutritional status in male athletes and controls. *J. Am. Coll. Nutr.* 11:181-191, 1992.
12. Fogelholm, M., J. Laakson, J. Lehto, and I. Ruokonen. Dietary intake and indicators of magnesium and zinc status in male athletes. *Nutr. Res.* 11:1111-1118, 1991.
13. Golf, S.W., D. Bohmer, and P.E. Nowacki. Is magnesium a limiting factor in competitive exercise? A summary of relevant scientific data. In *Magnesium 1993*, S. Golf, D. Dralle, and L. Vecchiet (Eds.). London: John Libbey & Company, 1993, pp. 209-220.
14. Golf, S., V. Graef, H.-J. Gerlach, and K.E. Seim. Veranderungen der serum-CK- und serum-CK-MB-aktivitaten in abhangigkeit von einer magnesiumsubstitution bei leistungssportlerinnen. *Magnesium Bull.* 2:43-46, 1983.
15. Goodwin, J.S., W.C. Hunt, P. Hooper, and P.J. Garry. Relationship between zinc intake, physical activity, and blood levels of high-density lipoprotein cholesterol in a healthy elderly population. *Metabolism* 34:519-523, 1985.
16. Haralambie, G. Serum zinc in athletes during training. *Int. J. Sports Med.* 2:135-138, 1981.
17. Haralambie, G., and J. Keul. The response of serum ceruloplasmin and copper during prolonged athletic training. *Arztl. Forschg.* 24:112-115, 1970.
18. Hickson, J.F., J. Schrader, and L.C. Trischler. Dietary intakes of female basketball and gymnastics athletes. *J. Am. Diet. Assoc.* 86:251-254, 1986.
19. Hooper, P.L., L. Visconti, P.J. Garry, and G.E. Johnson. Zinc lowers high-density lipoprotein cholesterol levels. *J.A.M.A.* 244:1960-1961, 1980.
20. International Commission on Radiological Protection. *Report of the Task Group on Reference Man* (I.C.R.P. Report No. 23). London: Pergamon Press, 1975, pp. 327-328.
21. Jacob, R.A., H.H. Sandstead, J.M. Munoz, L.M. Klevay, and D.B. Milne. Whole-body surface loss of trace metals in normal men. *Am. J. Clin. Nutr.* 34:1379-1383, 1981.
22. Krotkiewski, M., M. Gudmundsson, P. Backsrtrom, and K. Mandroukas. Zinc and muscle strength and endurance. *Acta Physiol. Scand.* 116:309-311, 1982.
23. Liu, L., G. Borowski, and L.I. Rose. Hypomagnesemia in a tennis player. *Phys. Sportsmed.* 11:79-80, 1983.
24. Lukaski, H.C. Interactions among indices of mineral element nutriture and physical performance of swimmers. In *Sports Nutrition: Minerals and Electrolytes*, J. Driskell and C. Kies (Eds.). Boca Raton, FL: CRC Press, 1995, pp. 265-277.

25. Lukaski, H.C. Prevention and treatment of magnesium deficiency in athletes. In *Magnesium and Physical Activity*, L. Vecchiet (Ed.). Carnforth, UK: Parthenon, 1995, pp. 211-226.
26. Lukaski, H.C., W.W. Bolonchuk, L.M. Klevay, D.B. Milne, and H.H. Sandstead. Maximal oxygen consumption as related to magnesium, copper and zinc nutriture. *Am. J. Clin. Nutr.* 37:407-415, 1983.
27. Lukaski, H.C., B.S. Hoverson, S.K. Gallagher, and W.W. Bolonchuk. Physical training and copper, iron and zinc status of swimmers. *Am. J. Clin. Nutr.* 53:1093-1099, 1990.
28. Mason, K.S. A conspectus of research on copper metabolism and requirements of man. *J. Nutr.* 109:1979-2066, 1979.
29. Shils, M.E. Magnesium. In *Modern Nutrition in Health and Disease* (8th ed.), M.E. Shils, J.A. Olson, and M. Shike (Eds.). Philadelphia: Lea & Febiger, 1993, pp. 164-184.
30. Singh, A., P.A. Deuster, and P.B. Moser. Zinc and copper status of women by physical activity and menstrual status. *J. Sports Med. Phys. Fitness* 30:29-35, 1990.
31. Solomons, N.W. On the assessment of zinc and copper nutriture in man. *Am. J. Clin. Nutr.* 32:865-871, 1979.
32. Spencer, H. Minerals and mineral interactions in human beings. *J. Am. Diet. Assoc.* 86:864-867, 1986.
33. Stendig-Lindberg, G., Y. Shapiro, Y. Epstein, E. Galun, E. Schonberger, E. Graff, and W.E. Wacker. Changes in serum magnesium concentration after strenuous exercise. *J. Am. Coll. Nutr.* 6:35-40, 1987.
34. Vallee, B.L., and K.H. Falchuk. The biochemical basis of zinc physiology. *Physiol. Rev.* 73:79-118, 1993.
35. Van den Hamer, C.J.A., T.U. Hoogeraad, and E.R.K. Klompjan. Persistence of the antagonistic influence of zinc on copper absorption after cessation of zinc supplementation for more than five days. *Biol. Trace Elem. Res.* 1:99-106, 1984.
36. Weight, L.M., K.H. Myburgh, and T.D. Noakes. Vitamin and mineral supplementation: Effect on the running performance of trained athletes. *Am. J. Clin. Nutr.* 47:192-195, 1988.
37. Weight, L.M., T.D. Noakes, D. Labadarios, J. Graves, P. Jacobs, and P.A. Berman. Vitamin and mineral status of trained athletes including the effects of supplementation. *Am. J. Clin. Nutr.* 47:186-191, 1988.

Author Notes

U.S. Department of Agriculture, Agricultural Research Service, Northern Plains Area, is an equal opportunity/affirmative action employer, and all agency services are available without discrimination.

Mention of a trademark or proprietary product does not constitute a guarantee or warranty of the product by the United States Department of Agriculture and does not imply its approval to the exclusion of other products that may also be suitable.

A reprint from

INTERNATIONAL JOURNAL OF

SPORT NUTRITION

HUMAN KINETICS PUBLISHERS